Phosphoric acid, tris(2-methylpropyl) ester: Human health tier II assessment

12 December 2019

CAS Number: 126-71-6

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted

and published separately, using information available at the time, and may be undertaken at different tiers.

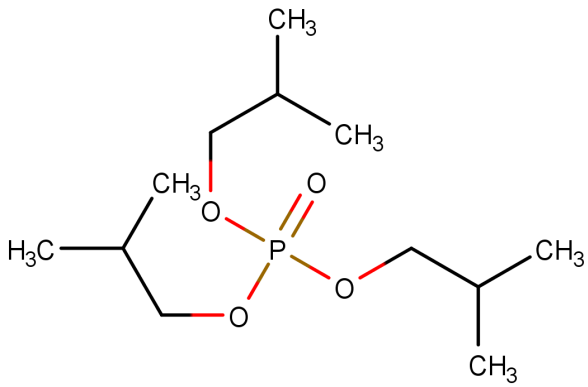
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:www.nicnas.gov.au

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	triisobutyl phosphate (TiBP) phosphoric acid, triisobutyl ester (8CI) isobutyl phosphate
Structural Formula	
Molecular Formula	C12H27O4P
Molecular Weight (g/mol)	266.31
Appearance and Odour (where available)	Colourless liquid
SMILES	C(C)(C)COP(=O)(OCC(C)C)OCC(C)C

Import, Manufacture and Use

Australian

No specific Australian use, import, or manufacturing information has been identified.

International

The following international uses have been identified through: the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers; Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (CosIng) database; the US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR), the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and various international assessments including from the Agency for Toxic Substances and Disease Registry (ATSDR).

The chemical is predominantly used in industrial plasticisers, polymers, rubbers, plastics, vinyl resins, and flame retardants (ATSDR, 2012).

The chemical has reported domestic uses, including in:

- cleaning/washing agents;
- surface active agents; and
- paints, lacquers and varnishes.

The chemical may also be used in hobby products such as modelling clay and finger paint.

The chemical is not listed in the US Household Products database (US HPD) or the American Cleaning Institute (ACI) database, indicating that domestic use of the chemical may not be widespread.

The chemical has reported domestic uses in the SPIN database. However, it should be noted that SPIN does not distinguish between direct use of the chemical, or use of the materials that are produced from chemical reactions involving the chemical.

The chemical has reported commercial uses, including:

- in surface coatings and adhesives;
- in automotive care products;
- in leather treatment products; and
- as an antifoaming agent.

The chemical has reported site-limited uses, including:

- in construction materials;
- as process regulators; and
- as a solvent for the extraction of metals.

Restrictions

Australian

No known restrictions have been identified.

International

No known restrictions have been identified.

Existing Work Health and Safety Controls

Hazard Classification

The chemical is not listed on the Hazardous Chemical Information System (HCIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available for the chemical. However, the following exposure standards are identified for 'tributyl phosphate, all isomers' (Galleria Chemica):

- time weighted average (TWA) of 5 mg/m^3 and
- short-term exposure limit (STEL) of 5 mg/m^3 in Ireland, South Africa and United Kingdom.

Health Hazard Information

Toxicokinetics

No data are available on the chemical.

Based on the available data for other phosphate esters, including tributyl phosphate (TBP), the chemical is expected to be readily absorbed after ingestion through the gastrointestinal tract (GIT), and to some extent after dermal or inhalation exposure (ATSDR, 2012). Studies with several phosphate esters have shown that these substances can be readily absorbed through the GIT, possibly via passive diffusion.

Dermal absorption can occur and will depend on the alkyl chain length. Shorter chain compounds are absorbed better than long chain compounds (ATSDR, 2012). Phosphate esters including TBP and (tris(1,3-dichloro-2-propyl) phosphate) TDCP were shown to be absorbed in significant amounts through rat skin, but TBP was poorly absorbed through pig skin (ATSDR, 2012; NICNASa).

Urinary excretion is reported to be the main route of elimination for some phosphate esters (ATSDR, 2012).

Acute Toxicity

Oral

The chemical has low acute toxicity based on results from animal tests following oral exposure. The median lethal dose (LD50) in rats is >3000 mg/kg bw.

In a guideline acute oral toxicity study following EPA OPP 81-1, the chemical was administered to Sprague Dawley rats (5/sex) in a single gavage dose of 5000 mg/kg bw. Reported clinical signs of toxicity included oral and nasal discharge, wet rales, urinary and faecal staining, hypoactivity, abdominal griping. One mortality occured following administration of the chemical. For this animal, examinations showed some changes in the lungs and gastrointestinal tract, indicating potential irritant effects. Two other animals exhibited dilated renal pelvis and/or pelvis containing white fluid. No other signs of toxicity were reported. The LD50 in this study was >5000 mg/kg bw (REACH).

Other reported LD50 values are (BG RCI, 2000; REACH):

- >4180 mg/kg bw in rats;
- 3200–6400 mg/kg bw in rats (clinical signs included ataxia, jerking and white foam);
- 6400–12800 mg/kg bw in mice (clinical signs included ataxia);
- 3072–6144 mg/kg bw in rats.

Dermal

The chemical has low acute toxicity based on results from animal tests following dermal exposure. The LD50 in rabbits is >5000 mg/kg bw.

In a guideline acute dermal toxicity study following EPA OPP 81-2, New Zealand White (NZW) rabbits (n = 5/sex/dose) were exposed to the chemical at 5000 mg/kg bw under an occlusive patch for 24 hours. No mortality occured following exposure. Reported clinical signs of toxicity included severe dermal effects (necrosis, eschar formation and exfoliation of eschar tissues), nasal discharge, urinary and faecal staining. One animal had clear fluid in the abdominal cavity, but no other signs of toxicity were reported. The LD50 in this study was >5000 mg/kg bw (REACH).

An LD50 >9600 mg/kg bw was reported for guinea pigs (REACH).

Inhalation

The chemical has low acute toxicity based on results from animal tests following inhalation exposure. The median lethal concentration (LC50) in rats is >5.14 mg/L.

In an acute inhalation toxicity study following OECD TG 403, Wistar rats (n = 5/sex/dose) were exposed (nose only) to the chemical at 5.14 mg/L (aerosol) for 4 hours. No mortality occurred following exposure. Reported clinical signs of toxicity included irregular breathing, gasping, tremors, uncoordinated and staggering gait, decreased spontaneous activity, ruffled fur, squatting, retracted flanks, reduced reflexes, blood-coloured crusted noses, sneezing, narrowed palpebral fissures, red-stained saliva and nasal discharge and swollen belly. The LC50 was >5.14 mg/L in this study (BG RCI, 2000; REACH).

No mortality was observed in rats, rabbits or cats exposed to 1.7 mg/L TiBP vapour for 6 hours. Mice exposed to the same concentration were more sensitive (8/10 surived a 6 hour exposure) (BG RCI, 2000).

Corrosion / Irritation

Skin Irritation

The chemical is slightly irritating to skin based on animal data.

In a skin irritation study following OECD TG 404, NZW rabbits (n = 3) were exposed to the undiluted chemical in semi-occlusive patch for 4 hours. Maximum erythema score of 1 (in 2/3 animals) and oedema score of 0 (in 3/3 animals) were reported for the 24, 48 and 72 hours observation times. Erythema scored 1 (very slight erythema) was fully reversible within 7 days. No clinical signs of toxicity were reported. The chemical was only slightly irritating to skin in this study (REACH).

In a non-guideline dermal irritation study, the chemical was tested in rabbits at concentrations of 20 or 50 % in oil applied on the back or the ears. Exposure duration and number of animals were not indicated. The low concentration was reported to produced minor irritation while the high concentration was reported to produced slight irritation on the back and ears, with haemorrhagic and necrotic inflammation (REACH).

Eye Irritation

The chemical is slightly irritating to eyes based on animal data.

In a eye irritation study following EPA OPP 81-4, the chemical was instilled into the lower conjunctival sac of the right eye of NZW rabbits (n = 6) for 24 hours. Reported effects were mild to moderate transient ocular irritation. Most severe effects were reported at 1 hour post-application, primarily redness, chemosis and discharge, and in one animal iridial changes. No corneal changes were reported. Effects were reported to be fully reversed within 24 hours for 4 rabbits, and within 48–72 hours for the remaining 2 rabbits (REACH).

Sensitisation

Skin Sensitisation

The chemical is not expected to be a potent skin sensitiser based on the weight of evidence including:

- a chemical structure that indicate low chemical reactivity;
- a negative result in the direct peptide reactivity assay (DPRA) (OCED TG 442C);
- a negative result in a keratinocyte activation test (OECD TG 442D);
- negative skin sensitisation data for structurally similar chemicals; and
- mixed results in guinea pig guideline studies (OECD TG 406).

In a maximisation test (adjuvant test method) following OECD TG 406, the chemical (no indication of purity) was found sensitising when tested in Pirbright guinea pigs (n = 10). For induction, the chemical was tested at 0.05 % and 4 % via intradermal route and epicutaneous routes, respectively. For challenge, animals were topically exposed to the chemical at 4 %. Positive reactions were observed in 9/10 treated animals compared with none in the control group (n = 5). No details on the skin reactions were provided. The chemical was reported to be sensitising (BG RCI, 2000; REACH).

In a Buehler test (non-adjuvant test method) following OECD TG 406, treatment with the chemical (99.2 % pure) was found sensitising when tested in Pirbright guinea pigs (n = 20). For induction, animals were exposed to topical applications of the chemical at 75 % for 6 hours on day 0, 7 and 14. For challenge, animals were exposed topically to the chemical at 50 %, 14 days after last induction (REACH). Very slight to well-defined skin reactions were observed in 9/18 animals treated with the chemical, compared with no reactions observed in controls (n = 10) (BG RCI, 2000; REACH).

In Buehler test following OECD TG 406, the chemical (no indication of purity) produced negative results in male Hartley guinea pigs (n = 10). For induction, animals were exposed 3 times to topical applications of the undiluted chemical for 6 hours. For challenge, animals were exposed topically to the undiluted chemical, 14 days after last induction. None of the treated animals showed skin reactions, whereas the positive control elicited reactions in all treated animals (n = 10). The chemical was reported to be non-sensitising in this study (BG RCI, 2000).

Structural analogue information

The structurally similar chemicals tributyl phosphate (TBP) and tri-(2-chloroisopropyl)phosphate (TCPP) were reported negative for skin sensitisation in guideline studies OECD TG 406 (Buehler test) and OECD TG 429 (LLNA), respectively (NICNASa; NICNASb).

In vitro

The chemical was negative in the in vitro assays for the first two key events (KE) in the adverse outcome pathway (AOP) for skin sensitisation. Importantly, the chemical was negative for the key initiating event 1 (protein binding).

Three in vitro studies were undertaken. Each of these guideline studies represents a key event of the AOP for skin sensitisation including: protein binding to form a hapten (OECD TG 442C); induction of antioxidant genes in keratinocytes (skin cells) (OECD TG 442D); and activation of a monocytes (immune cells) (OECD TG 442E).

- The chemical was reported negative in a direct peptide reactivity assay (DPRA) similar to OECD TG 442C. When tested at a concentration of 3.76 % in acetonitrile, the chemical did not show significant reactivity with either lysine or cysteine peptides (REACH).
- The chemical was reported negative in a keratinocyte activation test (LuSens assay) similar to OECD TG 442D. When tested in 2 experiments in 1 % DMSO (dimethyl sulfoxide) on the keratinocyte cell line LuSens for 48 hours at concentrations of 22.85–81.81 µg/mL (experiment 1) and 27.42–98.24 µg/mL (experiment 2), the chemical did not induce a significant luciferase activity as fold induction remained <1.5 in both experiments (REACH).
- The chemical was reported positive in a human cell line activation test (h-CLAT) similar to OECD TG 442E. When tested on the human cell line THP-1 at concentrations of 25–89 µg/mL for 24 hours, the chemical induced dendritic CD54 expression (REACH).

Quantitative Structure-Activity Relationship (QSAR) information

The QSAR modelling for skin sensitisation using the OECD QSAR Toolbox (version 4.2) indicated that there was only one structural alert for protein binding via nucleophilic substitution for the parent chemical and one metabolite (skin metabolism). Modelling did not give alerts for sensitisation via autoxidation.

Modelling using the knowledge-based expert system Deductive Estimation of Risk from Existing Knowledge (DEREK) Nexus 6.0.1 indicated that TiBP may be a weak sensitiser with the estimated concentration needed to produce a three-fold increased in lymphocyte proliferation (EC3) value of 100 %. The prediction contained an alert for alkyl esters from phosphoric or phosphonic acid. Phosphate esters contain two sites that can potentially react with proteins to form haptens. The phosphorus centre can react with hard nucleophiles (e.g. lysine amino groups). The alkyl group can react primarily with soft nucleophiles (e.g. cysteine thiol groups); however, phosphate esters are generally not very potent alkylating agents (DEREK 6.0.1). These factors are not expected to vary greatly between TiBP and the structurally similar chemical TBP.

The clearest chemical difference between TBP and TiBP is the presence of a tertiary carbon atom in the latter compound. This is suggestive that sensitisation (from TiBP) most likely arises from peroxidation at this more reactive site, leading to differences between samples based on their history.

Repeated Dose Toxicity

Oral

Based on the available data, the chemical is not expected to be harmful following repeated oral exposure.

In a subchronic oral toxicity study following OECD TG 408, the chemical was administered to SD rats (10/sex/dose) at concentrations of 0, 200, 1000 or 5000 ppm in the diet for 13 weeks (equivalent to 0, 13.9, 68.4 and 346.1 mg/kg bw/day for males and 0, 16.8, 84.3 and 403.9 mg/kg bw/day for females). Additional groups of rats (10/sex/dose) were used as control and high dose levels for histological examination of the bladder (BG RCI, 2000; REACH). No mortality occured during the study. No clinical signs of toxicity were reported. Histology of organs (including bladder) was normal. Reported observed effects included significantly decreased neutrophil count in high-dose males, significantly increased mean corpuscular haemoglobin concentration in mid- and high-dose males, and statistically significant increase in cholesterol in high-dose males. A non-significant decrease in neutrophil count was observed in high-dose females. The reported no observed effect level (NOEL) was

403.9 mg/kg bw/day for females based on the absence of toxicologically relevant effects and 68.4 mg/kg bw/day for males, based on significantly decreased neutrophil count at the highest dose (BG RCI, 2000; REACH).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

Based on the weight of evidence from the available in vitro and in vivo genotoxicity studies, the chemical is not considered to be genotoxic.

In vitro

The chemical was tested and found negative in the following in vitro studies:

- a bacterial gene mutation assay following OECD TG 471 in *Salmonella typhimurium* strains TA1535, TA1537, TA98 and TA100, exposed to concentrations up to 5000 µg/plate with and without metabolic activation (REACH);
- bacterial gene mutation assay in *S. typhimurium* strains TA1535, TA1537, TA1538, TA98 and TA100, exposed to concentrations up to 5000 µg/plate with and without metabolic activation (REACH);
- bacterial gene mutation assay in *S. typhimurium* strains TA1535, TA1537, TA98 and TA100, exposed to concentrations up to 1500 µg/plate with and without metabolic activation (REACH);
- a mammalian cell gene mutation study following OECD TG 476 in Chinese hamster lung fibroblasts (V79) exposed to concentrations up to 2660 µg/mL (in the range-finding study), with and without metabolic activation.

In vivo

In a micronucleus test following EPA OPPTS 870.5395, the chemical was administered as a single intraperitoneal dose of 0, 300, 600 or 1200 mg/kg bw to CD-1 mice (n = 15/sex/dose). No mortality occured following exposure. No clinical signs of toxicity were reported at any of the doses tested. No significant increases in micronucleated polychromatic erythrocyte (PCE) frequency were reported (BG RCI, 2000; REACH).

Carcinogenicity

No data are available for the chemical.

The structurally similar chemical TBP caused bladder cancer in rats which was preceded by bladder irritation and adverse histopathological findings. However, TiBP did not cause bladder irritation or adverse histopathlogy in rats (see **Repeat dose toxicity**).

Reproductive and Developmental Toxicity

No data on effects on fertility are available. Based on the available information, the chemical is not expected to be toxic to foetal development. Although skeletal malformations were observed in rat foetuses at doses starting at 100 mg/kg bw/day, in particular at the highest dose of 1000 mg/kg bw/day, no treatment-related developmental effects were reported in rabbits at doses up to 150 mg/kg bw/day.

In a prenatal developmental toxicity study following OECD TG 414, the chemical was administered by gavage at doses of 0, 100, 300 or 1000 mg/kg bw/day to pregnant Charles River rats (25/dose) during gestation days (GD) 6–15. No mortality occured during the study. Clinical signs of maternal toxicity included salivation (occasionally brown-stained) and wet fur in all animals at mid the and high doses, and in a lesser number at the low dose. At the highest dose, a slight and temporary retarded body weight gain was reported between initiation and GD 8. Water but not food consumption was reported to be increased at the highest dose. A marginal increase in early embryonic losses was reported at the highest dose, with no statistical significance. Incidences of pre and post implantation losses were not affected by the treatment. A NOAEL of 300 mg/kg bw/day was reported for maternal toxicity. Foetal body weight, sex ratio and litter size were reported to be unaffected by treatment. Skeletal malformations were reported in 2, 3 and 8 foetuses at low, mid and high dose, respectively, compared with 2 foetuses in the control group. At high dose, 6 foetuses had bilateral forelimb flexure, associated with distorted rib cage and thickened ribs in 5 foetuses. There was no consistent pattern in the other types of malformation observed and no other related adverse effects were observed. Therefore, the slight increase in malformations was considered unlikely to be treatment-related. Based on these observations, a NOAEL of 300 mg/kg bw/day for developmental toxicity can be derived (BG RCI, 2000; REACH).

In a prenatal developmental toxicity study following OECD TG 414, the chemical was administered by gavage at doses of 15, 50 or 150 mg/kg bw/day to NZW female rabbits (22/dose) on days 6–28 post-coitum (REACH). One rabbit of the high dose group was euthanised on day 23 after presenting persistent body weight loss, severely reduced food consumption, reduced faeces production and piloerection. One mortality occured on on day 28, possibly due to oral misgavage. Moderate body weight loss was observed in some animals at 50 and 150 mg/kg bw/day, with no dose-response relationship. Across all treated groups, slight body weight loss was reported, along with lower relative food consumption. Reduced faeces production (up to severe) was reported in all groups including control, with no clear dose-response relationship. No other mortality and treatment-related maternal signs of toxicity were reported. At mid dose, the mean incidence of preimplantation loss was reported to be higher (15.9 %) than controls (5.2 %), and slightly higher than historical controls (15.3 %), but was considered incidental in the absence of dose-dependent and statistically significant effects at higher doses. Mean number of corpora lutea and implantation sites were reported to be within the range of historical control data. A total of 5 animals were not pregnant in the study, the others all had litters with viable foetuses. A maternal NOAEL of 150 mg/kg bw/day was determined based on the absence of toxicologically relevant maternal effects. No significant developmental effects were reported. The reported number of external, skeletal and visceral malformations in foetuses from treated groups was not significantly different from the control group. A NOAEL of 150 mg/kg bw/day for developmental toxicity was found in this study (BG RCI, 2000; REACH).

Other Health Effects

Neurotoxicity

Neurotoxicity is a potential adverse effect of many organophosphates, but the potency levels that may cause neurotoxicity varies significantly. Based on the available data, the chemical is not expected to be neurotoxic.

In a neurotoxicity study following OECD TG 418, the chemical was orally administered to groups of hens (5/dose) in a single dose of 0, 500, 1000, 2000 or 5000 mg/kg bw. The groups receiving a single dose of 2000 and 5000 mg/kg bw had a second dose 21 days following the first administration. No mortality and no treatment-related effects were reported in treated hens, whereas 5/15 animals died in the positive control [tri-ortho-cresyl phosphate (TOCP)] group and 7/15 positive control animals exhibited severe signs of neurotoxicity (BG RCI, 2000; REACH).

In a neurotoxicity study following OECD TG 418, the chemical was orally administered to white Leghorn hens (10) at a single dose of 5000 mg/kg bw. Negative and positive control (TOCP) groups of 5 hens were used. Enzymatic activity was measured 24 hours after administration and showed that plasma butyrylcholinesterase was inhibited in treated hens. Brain acetylcholinesterase (AChE) and neurotoxic esterases (NTE) activities were not impacted by treatment, suggesting that the chemical did not have delayed neurotoxic effects in hens (BG RCI, 2000; REACH).

Risk Characterisation

Critical Health Effects

While no classifiable hazards have been identified, the chemical may cause skin sensitisation and prolonged exposure to the chemical may cause skin irritation.

Public Risk Characterisation

Although use in domestic products in Australia is not known, the chemical is reported to be used in domestic products overseas. The data indicate that the majority of uses in domestic products that could expose the public directly to the chemical, is not frequent or widespread. However, the chemical has reported international use in hobby products including finger paint and modelling clay (where intentional skin contact is expected). Prolonged exposure of these hobby products to air may lead to formation oxidation products with the ability to bind proteins (see **Skin sensitisation**). While this poses a risk in theory, no case reports have been identified, indicating that the actual risk is small to non-existent. Therefore, the risk to the public posed by domestic products including hobby products containing the chemical is not considered to be unreasonable.

The public may be directly exposed via articles or coated surfaces containing the chemical. While flame retardants are retained well in intact articles, there is potential for them to be released if the article breaks down into smaller pieces or dust. The chemical could be released from articles through abrasion or dissolution (ATSDR, 2012). However, the use of the chemical in articles in Australia is unknown.

The chemical is sometimes detected in house dust but only at low levels (Brandsma et al., 2014; Van den Eede et al., 2011) Therefore, the exposure to the chemical from this source is expected to be low.

Should further information to better characterise exposure to TiBP become available, further assessment may be required.

Occupational Risk Characterisation

During product formulation, dermal and inhalation exposure might occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemical at lower concentrations could also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the local health effects, the chemicals could pose an unreasonable risk to workers unless adequate control measures to minimise repeated or prolonged dermal exposure are implemented.

The chemical currently has no hazard classification for worker health and safety; this is considered appropriate based on the available data.

NICNAS Recommendation

Current risk management measures are considered adequate to protect public and workers' health and safety, provided that all requirements are met under workplace health and safety, and poisons legislation as adopted by the relevant state or territory.

Regulatory Control

Work Health and Safety

The chemical is not recommended for classification and labelling aligned with the Globally Harmonized System of Classification and Labelling of Chemicals (GHS). This does not consider classification of physical hazards and environmental hazards.

Advice for industry

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemical is prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the Preparation of safety data sheets for hazardous chemicals—Code of practice and Labelling of workplace hazardous chemicals—Code of practice, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of the chemical has not been undertaken as part of this assessment.

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